Calcium and Phosphorus for Cats and Dogs

Daniel C. Barrette

Calcium and phosphorus are the two minerals cats and dogs need most. Particularly in dogs, growth is phenomenal; small breeds reach their adult weight in 9 to 12 months, and large breeds attain 90 to 95% of their adult weight in 12 to 15 months. Growth is particularly rapid in the first six months, when the birth weight increases 40 to 50 times. It is, therefore, essential to control both these important minerals.

Physiological Roles

Calcium's role in building strong bones and teeth is well known. It also plays an active part in blood clotting, transmission of nerve impulses, and secretory and membranous activities; it is a vital mineral. Phosphorus, on the other hand, is involved in more metabolic functions than any other mineral: bone formation, energy metabolism, membrane integrity, nucleic acid metabolism, buffer action, etc.

The body as a whole consists of 0.7% minerals, primarily macrominerals: calcium, phosphorus, potassium, salt, magnesium and sulphur. It is, however, estimated that to meet dogs' mineral requirements, 4 to 5% of the dry diet material should be ash. All nutritional minerals are not completely available, so their proportion in the diet must be increased. But commercial dry food contains 7 to 9% ash. Although these levels are not necessarily harmful, they frequently indicate that quality has been compromised.

Physiological Controls

Half of blood calcium is ionized (Ca++). The rest is bonded to phosphates or proteins. If the serum protein rate increases, so does serum calcium. The body attempts to maintain its ionized calcium level in the blood; it will succeed in two ways: by fostering the absorption of intestinal calcium, and stimulating the reabsorption of calcium by the bones, in the event of low-level calcemia, and in the opposite case, by fostering calcium deposit in the bones and renal excretion of the mineral. Calcium losses also occur during lactation and pregnancy (fetal growth). And with biliary and pancreatic secretions, the body can return calcium to the intestine.

Low-level calcemia is caused primarily by a decrease in ionized calcium; when this occurs, the body secretes parathormone, which stimulates formation of 25-hydroxycholecalciferol by the liver and 1,25-diOH-cholecalciferol (DOCC) by the kidneys. This latter vitamin D3 metabolite acts like a real hormone, fostering intestinal absorption of calcium in several ways. In addition, with the help of the parathormone, DOCC stimulates calcium mobilization by bone reabsorption, increasing the number and activity of osteoclasts.

The parathormone also acts on the renal tubule. First it inhibits the reabsorption of tubular phosphate; the result is increased phosphate in the urine. This inhibits the increase of phosphatemia, which, in any case, is the result of reabsorption by the bones. The parathormone also increases the tubular reabsorption of calcium to help restore ionized plasma calcium; however, this action is not very valuable in dogs and cats, given the small quantities of calcium their kidneys excrete.

On the other hand, with elevated calcemia, the parathormone is somewhat inactive; the most active hormone at that juncture is calcitonin, which reduces osteoclastic activity, thus decreasing new plasma calcium levels, reversing the parathormone action.

Control measures for phosphoremia are much less precise than for calcemia. Serum phosphorus varies enormously in terms of the assimilation of dietary phosphorus, which is not so much the case with calcium.

Calcium and Phosphorus Nutritional Requirements

Nutritional requirements may be expressed in several ways; as a percentage of dry diet or serving, as a quantity per animal per day, per 1000 calories metabolized, etc. The first method is the most popular with veterinarians. According to the 1985 American National Research Council (NRC) standards for dogs, the minimum dry diet requirements for growth are 0.59% assimilable calcium and 0.44% assimilable phosphorus. However, the diet must take into account the bioavailability of these minerals. That is why most authors recommend that dry food contain 1.1% calcium and 0.9% phosphorus. During growth and lactation, Lewis recommends 1.0 to 1.8% calcium and 0.8% to 1.6% phosphorus. During late gestation and growth, he

Daniel Barrette, D.M.V., member of the Nutrition Subcommittee of the CVMA Pet Food Certification Program, and currently professor in the Department of Anatomy and Physiology, Faculté de Médecine vétérinaire, Université de Montréal.

NDLR: Cet article a été publié en français en janvier/février 1987.

advises 0.8 to 1.5% calcium and 0.6 to 1.2% phosphorus.

In addition to levels, it is essential that the ratio between the two minerals be observed. Researchers recommend an optimum Ca:P ratio of 1.2 to 1.4:1. Some say this ratio could rise to 2:1, but it should never drop below 1:1.

For cats, the most recent NRC recommendations (1978) set dry diet requirements at 1.0% calcium and 0.8% phosphorus. In other words, cats need 200 to 400 milligrams of calcium a day for bone development, and adult cats have 40 grams of calcium in their bones. The optimum ratio of available calcium:phosphorus should be between 0.9 and 1.1:1, but since phosphorus, particularly grain-source phosphorus, is partially bonded to phytic acid, phosphorus assimilation is proportionally decreased if there is a great deal of grain in the diet. The actual dietary Ca:P ratio should be approximately 1.1 to 1.4:1.

In popular food stuffs often fed to canines and felines, the Ca:P ratio is generally unfavorable: 1:50 in liver, 1:40 in heart, 1:10 in meat, 1:5 in rice and 1:4 in bread. So homeprepared food must be corrected. For example, to restore the appropriate Ca:P ratio, 400 mg of calcium must be added to the daily diet of a dog fed only meat.

The Dangers of Imbalance

It is essential for animal health that provision be made for not only the quantities of calcium and phosphorus required for development, but also of the ratio between the two elements.

In dogs, an unbalanced all-meat diet leads to osteofibrosis. This type of diet also contains an aggravating factor: because of the protein excess, surplus sulphur amino acids are excreted in the form of calcium sulphate. Not only is the Ca:P ratio deficient, but the urinary excretion of calcium is exaggerated, which accelerates the process of the illness.

The primary symptoms of osteofibrosis are anxiety and anorexia, particularly in puppies, diarrhea, painful skeletal deformation, which results in lameness, postural abnormalities and plantigrade gait. The hair coat is dull and dry. If bone dimineralization is pronounced, spontaneous green-stick fractures occur. In adults, there is parodontitis (abscesses, periodontal osteomyelitis, loose teeth). Because of the high level of urinary phosphates, phosphate urolithiases are also observed.

In cats, the same illness presents almost the same clinical symptoms. Siamese are particularly sensitive.

Treatment of an animal afflicted with osteofibrosis (osteodystrophy) involves a change in diet to a Ca:P ratio of 2:1. Milk is a good food if the animal will take it. A large, boiled veal bone may also be given, but it will cause intestinal obstruction if eaten. Chicken and rabbit bones must be avoided because of their dangerous fragility.

The other, more frequent, problem is excess calcium in the diet. It is very tempting for breeders and owners of large dogs to give them calcium supplements, notably because of poorly understood notions of calcium's role in growth. The first consequence of this addition is increasing calcemia. To counter this hypercalcemia, the body excretes calcitonin on a continual basis. We have seen that calcitonin reduces reabsorption by the bones. If the suppression of reabsorption is chronic, because of elevated calcitoninemia, the result will be a gradual thickening and increased density of the bones, leading to potential osteopetrosis. In a growing dog, however, the consequences are harmful; decreased bone reabsorption results in abnormal morphology of certain bone structures (acetabulum, head and neck of the femur, spinal column). These modifications lead to hip dysplasia and the wobbler syndrome. Excess dietary calcium (3 times the dog's requirement, in Hazewinkel's experiment) leads to delayed bone maturation and dissecting osteochondrosis. Furthermore, the long-term risk of soft tissue mineralization must also be kept in mind. In their attempt to display prudence and good will, breeders pitch their animals into the abyss they wanted to avoid. Veterinarians should advise their clients of the dangers of wanting to do too well.

Vitamin D overdoses are also dangerous. Researchers in Colorado (Stephens *et al*) compared two groups of correctly nourished dogs. The diet provided 1760 I.U. of vitamin D per kilo, four times the NRC recommendations. One group was kept inside; the other was exposed to ultraviolet solar radiation and received a supplement of 2.3 g of calcium per animal per day. These quantities of calcium, which are not extraordinary supplements, represented respective 12% and 84% increases over requirements for dogs kept inside and outside. Since phosphorus was not increased, there was an imbalance between the two minerals. The researchers demonstrated a resulting chronic hypercalcitoniemia which led to lowered calcemia in dogs exposed to U.V. solar radiation.

Deficiencies

Although they are rarer, the risks of deficiencies must not be neglected. Vitamin D, calcium and phosphorus deficiencies are responsible for osteomalacia in nursing females and rickets in the young. Calcium deficiency can still be observed in animals fed solely on (muscle) meat. Phosphorus deficiency is often secondary to diet that is very high in calcium, or results from a diet containing a great deal of grain, and therefore phytic acid. A diet that is high in lipids should also be supplemented with phosphorus; for each 3 to 20% increase in dietary fat, there should be a corresponding 20% increase in phosphorus.